Original Article

Role of Surface Electrocardiography in Differentiation between Obstructive and Non-Obstructive Hypertrophic Cardiomyopathy

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Abstract

Background: *Electrocardiography (ECG), as an easily accessible modality, is usually helpful in hypertrophic cardiomyopathy (HCM) diagnosis. The purpose of this study was to evaluate the role of ECG in differentiating between obstructive (OHCM) and non-obstructive (NOHCM) HCM.*

Methods: *The present study is a cross-sectional analysis of HCM patients referred to our center between 2008 and 2017. The study variables included age, sex, clinical presentation, medications, and ECG characteristics including PR interval, QRS width, QTc duration, Tpeak-Tend interval, QRS axis, QRS transition, ventricular hypertrophies, atrial abnormalities, ST-T abnormalities, and abnormal Q waves.*

Results: *The HCM sample consisted of 200 patients (55% males; age 45.60±15.50 y) from our HCM database. We compared the clinical and ECG characteristics of 143 NOHCM patients with those of 57 OHCM patients. The OHCM group was significantly younger than the NOHCM group (age =41.7 vs 47.0 y; P=0.016). The initial clinical presentation was similar between the 2 forms (P>0.05), and palpitations were the dominant symptom. Baseline ECG intervals, including PR (155.6 vs 157.9 ms), QRS (82.5 vs 82.0 ms), and QTc (430.5 vs 433.0 ms), were similar (all Ps>0.050). There were no differences regarding baseline rhythm, atrial abnormalities, QRS transition, ventricular hypertrophies, axis changes, ST-T changes, and abnormal Q waves between the HCM groups (all Ps>0.05).*

Conclusion: *The present study showed that standard 12-lead ECG had no role in distinguishing patients with the obstructive and non-obstructive forms of HCM.*

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Introduction

Hypertrophic cardiomyopathy (HCM) is a relatively common inherited disease that is the most common cause of sudden cardiac death in young adults.[1] HCM can present with dyspnea, palpitations, and less commonly syncope. However, it may be asymptomatic and only be diagnosed through routine investigations or an abnormal electrocardiogram (ECG) obtained for irrelevant causes.[2] Approximately, two-thirds of patients with HCM demonstrate a dynamic left ventricular outflow tract (LVOT) gradient at rest or with provocation.[3]

The diagnosis of HCM relies on the detection of increased LV wall thickness by imaging modalities, such as echocardiography and cardiovascular magnetic resonance. ECG remains a cornerstone in the assessment of patients with HCM, especially in differentiation from athlete's heart.[4] Even more, ECG is undergoing a revolution in the field of cardiomyopathies, not only because it is low price and enjoys widespread availability but also because it provides details related to morphology, function, and genetic substrates simultaneously.

LVOT obstruction is usually associated with some degree of mitral regurgitation, enhanced diastolic dysfunction, and aggravated myocardial ischemia.[5] We hypothesized that the presence of the LVOT gradient might produce different types of ECG features. It is noteworthy that the role of ECG in distinguishing obstructive (OHCM) from non-obstructive (NOHCM) HCM has not been studied adequately.[6] Therefore, we designed this study to evaluate the possible role of ECG in differentiating OHCM from NOHCM.

Methods

The initial study population was 246 patients, aged 18 to 80 years, with a definite HCM diagnosis between 2008 and 2017 at our center. After the initial screening, 46 patients were excluded. The exclusion criteria consisted of the presence of concomitant cardiac diseases, obstructive coronary artery diseases, a history of surgical reduction therapy or alcohol ablation, and significant right ventricular (RV) or LV systolic dysfunction. Finally, an HCM sample of 200 patients was enrolled in the study. This study was approved by the institutional ethics committee, and written informed consent was obtained from all subjects before inclusion in the study.

ECG tracings were recorded at the standard speed and amplification (25 mm/s, 10 mm=1 mV) and obtained at, or nearest to, the time of initial evaluation in all the individuals. All ECGs were independently analyzed by 2 experienced physicians (M.H. and A.F.), blinded to the final diagnosis, In the event of disagreement, a third physician was consulted (M.S-A).

ECG findings were defined according to the standard criteria[7]:

- *Ventricular hypertrophy:*
  1) Left ventricular hypertrophy (LVH) was defined as SV1+RV5 or RV6, whichever was larger, >35 mm (the Sokolow–Lyon index).
  2) Right ventricular hypertrophy (RVH) was defined as right axis deviation ≥ +110°, R wave in V1>7 mm or R/S ratio >1, dominant S wave in V5 or V6 >7 mm deep or R/S ratio <1, and QRS duration <120 ms.
- *Atrial abnormalities:*
  1) Left atrial abnormality was defined as prolonged P-wave duration >120 ms in leads I or II with a negative portion of the P-wave ≥1 mm in depth and ≥40 ms in duration in lead V1.
  2) Right atrial abnormality was defined as P-wave amplitude >2.5 mm in the inferior leads (II, III, and AVF) or >1.5 mm in V1 and V2.
- *Axis deviation:*
  1) normal axis: from −30° to +90°
  2) left axis deviation: from −30° to −90° on the frontal plane
  3) right axis deviation (RAD): from +90° to 180°
- *ST-T changes:*
  1) ST-segment depression (STD) >0.1 mV in depth in at least 2 adjacent leads
  2) ST-segment elevation (STE) >0.1m V in depth in at least 2 adjacent leads
  3) T-wave inversion (TWI) as negative T-waves >0.1 mV in at least 2 adjacent leads (except for aVR), in the absence of conduction disturbances
- pathological Q-waves: amplitude ≥25% of the ensuing R-wave and/or duration ≥0.04 s
- precordial transition: If the transition occurs at or before V2, it is called "an early transition". If the transition occurs after V4, it is called "a delayed transition". A normal transition occurs around V3 or V4.

ST-T changes and abnormal Q-waves were also categorized into 4 zones according to the recording ECG electrodes as inferior limb leads (II, III, and aVF), high lateral leads (I and aVL), right precordial leads (V1–V3), and left precordial leads (V4–V6). Tp-Te and QT intervals were measured in lead V5. If V5 was not suitable, leads V4 and V6 in that order were measured. QT interval was corrected for heart rate using the Bazett formula. Prolonged QTc was defined as QTc ≥470 ms in women and QTc ≥480 ms in men.

Transthoracic echocardiography was performed using a GE Vivid ultrasound machine (GE Ultrasound, Milwaukee, WI) with a multifrequency phased-array transducer. The diagnosis of HCM was based on the presence of a hypertrophied (wall thickness ≥15 mm) and non-dilated LV in the absence of other diseases that could produce the

same magnitude of hypertrophy.[2]

LVOT pressure gradients were measured in the apical views by continuous-wave Doppler echo under resting conditions and during provocative maneuvers, including Valsalva, treadmill or bicycle exercise, and/or amyl nitrite inhalation or dobutamine infusion, to elicit latent obstruction. After the measurement of peak resting and stress pressure gradients, the classification of HCM was established as NOHCM (<30 mmHg at rest and stress) and OHCM (≥30 mmHg at rest and stress).

The fitness of interval variables with a normal distribution was assessed using the 1-sample Kolmogorov–Smirnov test. Data were presented as the mean±the standard deviation for continuous and frequencies (percentages) for categorical variables. Comparisons of characteristics were made using the Pearson χ^2 test or the Fisher exact test for categorical variables and the Student *t* test for continuous variables. ECG characteristics between NOHCM and OHCM were compared using the independent sample *t* test. Independent predictors for HCM types were identified by logistic regression models. All parameters with P values ≤0.2 were entered into the logistic regression model. A P value <0.05 was considered statistically significant. The statistical analyses were performed using IBM SPSS Statistics 25 for Windows (IBM Corp, Armonk, NY).

Results

Two hundred patients, composed of 57 patients (28.5%) with OHCM and 143 patients (71.5%) with NOHCM, were included in the final analysis. Among all, 110 patients (55%) were men, and 90 (45%) were women. The patients were aged between 18 and 80 years, and the mean age was 45.60±15.50 years. Palpitations were the most common presenting symptom (64%). The mean symptom duration was 14 months (range= 2–120 mon).

ECG was abnormal in 96% (n=192) of the whole HCM population. Atrial fibrillation was the baseline rhythm in 11% (n=22) of the patients. One-third of the study population had an abnormal QRS axis (left axis deviation=51 [25.5%] and right axis deviation=10 [5%]). Atrial abnormality, mainly in the form of left atrial abnormality (n=102, 51%), was observed in 55% of the patients. The precordial transition was early in 41.5% of the studied patients (n=83), normal in 42% (n=84), and delayed in 16.5% (n=33). Isolated LVH and isolated RVH were present in 40% (n=80) and 5% (n=10) of the patients, respectively.

STD (n=111, 55.5%) and TWI (n=162, 81%) were the most common ECG abnormalities in our cohort. STD was chiefly observed in the left precordial (n=91, 45.5%) and high lateral limb (n=77, 38.5%) leads. STD was uncommon in the inferior limb (n=36, 18%) and right precordial (n=17, 8.5%) leads. Like STD, TWI was more commonly detected in the high lateral (n=134, 67%) and left precordial (n=122, 61%) leads than in the inferior (n=62, 31%) and right precordial (n=46, 23%) leads. Nonetheless, STE (n=17, 8.5%) and abnormal Q-wave (n=12, 6%) was an uncommon finding and was predominantly observed in the inferior limb leads (n=9, 4.5% and n=7, 3.5% respectively). Prolonged QTc was detected in 15% (n=30) of the patients.

From the clinical perspective, patients with the obstructive form of the disease were significantly younger at the time of diagnosis (mean age =41.7 y vs 47.1 y; *P*=0.016). Nevertheless, sex (*P*=0.870), clinical presentations (all *Ps*>0.05), and symptom duration (*P*=0.210) were similar. Similar antiarrhythmic medications (*P*=0.990) were administered for the 2 forms of HCM (Table 1).

Table 2 demonstrates detailed ECG characteristics in the OHCM and NOHCM groups. Baseline ECG intervals,

Table 1. Clinical presentations and medication history*

	Obstructive (n=57)	Non-obstructive (n=143)	*P*
Age	41.70±12.40	47.10±16.30	0.016
Sex			0.870
Male	32 (56.1)	78 (54.5)	
Female	25 (43.9)	65 (45.5)	
Symptoms			
Palpitations	38 (66.7)	90 (62.9)	0.630
Dizziness	16 (28.1)	56 (39.1)	0.610
Presyncope	4 (7.0)	12 (8.4)	0.790
Syncope	11 (19.3)	31 (21.7)	0.850
Symptom duration	12 (6.22)	8 (3.12)	0.210
Medications			
Beta-blockers	49 (86.0)	123 (86.0)	0.990
Calcium antagonists	6 (10.5)	15 (10.5)	0.990
Other AADs	2 (3.5)	5 (3.5)	0.990

*Data are presented as mean±SD or n (%).
AAD, Antiarrhythmic drugs

including PR interval (*P*=0.680), QRS width (*P*=0.950), QTc interval (*P*=0.560), and Tpeak-Tend (*P*=0.640), were comparable in OHCM and NOHCM. Atrial fibrillation was observed in 12% of the OHCM group and 10% of the NOHCM group (*P*=0.750). Although the majority of the patients in both groups showed normal QRS axis (OHCM =77.2% vs NOHCM =66.4%), there was a trend for more right axis deviation in the NOHCM group (OHCM =0% vs NOHCM =7%; *P*=0.087). LVH and RVH were seen in similar proportions of the patients in both HCM subtypes. ECG signs of atrial abnormalities were observed mainly in the form of left atrial abnormality (OHCM =43.9% vs NOHCM =53.8%; *P*=0.420). Most of the patients in the 2 HCM subtypes showed an abnormal precordial transition (OHCM =80.7% vs NOHCM =85.3%); however, QRS transition occurred with a similar pattern in the 2 groups (*P*=0.530). Although left precordial and high lateral limb leads were the most common locations for STD, the distribution of the STDs was similar in all lead zones (all *Ps*>0.050). Contrary to STD, STE was uncommon both in OHCM (1.8–3.5%) and NOHCM (0.7–3.5%). Still, both HCM groups showed a similar pattern of the STE distribution in 4 ECG zones (all *Ps*>0.050). TWI was the most common ST-T abnormality in both OHCM and NOHCM groups. High lateral limb leads and left precordial leads showed the highest prevalence of TWI. Nonetheless,

Table 2. Baseline electrocardiographic findings*

	Obstructive (n=57)	Non-obstructive (n=143)	*P*
PR interval	155.60±34.10	157.90±34.60	0.680
QRS width	82.50±24.50	82.20±23.0	0.950
Corrected QT interval	430.50±42.0	433.50±48.0	0.560
Tpeak-Tend interval	93.90±21.20	92.20±23.10	0.640
Baseline Rhythm			0.750
Sinus rhythm	50 (88.0)	128 (90.0)	
Atrial fibrillation	7 (12.0)	15 (10.0)	
Ventricular Hypertrophy			
Left ventricular hypertrophy	21 (37.0)	59 (41.0)	0.660
Right ventricular hypertrophy	13 (23.0)	30 (21.0)	0.700
Axis			0.087
Normal axis	44 (77.2)	95 (66.4)	
Left axis deviation	13 (22.8)	38 (26.6)	
Right axis deviation	0 (0.0)	10 (7.0)	
P-Wave Abnormality			0.420
Left atrial abnormality	25 (43.9)	77 (53.8)	
Right atrial abnormality	3 (5.3)	5 (3.5)	
Precordial Transition			0.530
Early transition	19 (33.3)	64 (44.7)	
Normal transition	11 (19.3)	21 (14.7)	
Late transition	27 (47.4)	58 (40.6)	
ST-Segment Depression			
Inferior leads	14 (24.6)	22 (15.4)	0.150
High lateral leads	25 (43.9)	52 (36.4)	0.320
Right precordial leads	3 (4.9)	14 (10.0)	0.230
Left precordial leads	29 (50.9)	62 (43.4)	0.330
ST-Segment Elevation			
Inferior leads	2 (3.5)	5 (3.5)	0.990
High lateral leads	2 (3.5)	2 (1.4)	0.330
Right precordial leads	1 (1.8)	1 (0.7)	0.490
Left precordial leads	2 (3.5)	1 (0.7)	0.140
T-Wave Inversion			
Inferior leads	20 (35.1)	42 (29.4)	0.430
High lateral leads	33 (57.9)	101 (70.6)	0.080
Right precordial leads	10 (17.5)	36 (25.2)	0.240
Left precordial leads	32 (56.1)	90 (62.9)	0.370
Abnormal Q-Wave			
Inferior leads	2 (3.5)	7 (4.9)	0.670
High lateral leads	1 (1.8)	2 (1.4)	0.850
Right precordial leads	2 (3.5)	3 (2.1)	0.560
Left precordial leads	2 (3.5)	8 (5.6)	0.540

*Data are presented as mean±SD or n (%).

Table 3. Multivariate analysis

	Odds ratio	95% confidence interval	*P*
Inferior lead ST depression	0.42	0.18-1.01	0.052
High lateral lead T inversion	1.28	0.64-2.56	0.480
Left precordial lead ST elevation	1.48	0.28-7.67	0.640

the distribution of TWI in all ECG zones was comparable in both groups (all *Ps*>0.05). Similar to STE, abnormal Q-waves were uncommon findings in both HCM subtypes with a similar distribution in 4 ECG zones (all *Ps*>0.05). A multivariate analysis using a binary logistic analysis was applied for the 4 parameters with a *P* value ≤0.2 (Table 3). We found no independent ECG predictor to distinguish between OHCM and NOHCM.

Discussion

To our knowledge, this is the first comprehensive study to evaluate possible differences in ECG findings between OHCM and NOHCM. Our results showed no benefits for ECG use in differentiating between OHCM and NOHCM.

Abnormal ECG was reported in more than 90% of the patients.[7] Although HCM is generally diagnosed by detecting LV wall thickness ≥15 mm on echocardiography or cardiovascular magnetic resonance, this degree of LVH is not specific to HCM and may be observed in other pathological conditions. In this setting, ECG is extremely useful in differentiating between HCM and its phenocopies. In the current study, 96% of the patients had abnormal ECGs, including atrial tachyarrhythmia, abnormal axis, atrial abnormality, abnormal precordial transition, abnormal ECG intervals, chamber hypertrophy, and ST-T abnormalities. Prior studies similarly reported an abnormal ECG in 90% to 96% of patients.[8-13] We think it is uncommon to find a completely normal ECG in patients with HCM documented by cardiac imaging. It is possible to observe normal ECGs in gene carriers without LVH. Although other ECG abnormalities, such as ST-T wave changes or signs of LVH, generally concur, isolated ECG signs of atrial abnormality may be observed in patients with HCM. Certain ECG abnormalities may precede the development of LVH in children, most commonly precordial voltages, and deep Q waves.[11] Hence, ECG is more sensitive than echocardiography as a screening tool in families with HCM.

We conducted a complete analysis of all possible ECG parameters and found no parameters that could differentiate between OHCM and NOHCM. In our cohort, significant QT prolongation was observed in 15% of patients with HCM, a prevalence that is significantly higher than that in healthy individuals. QTc prolongation likely reflects the interplay of cardiac hypertrophy, fibrosis, and electrophysiological remodeling of cardiomyocytes.[4] Johnson et al[14] reported that prolonged QTc was present in 1 out of 8 (12.5%) patients with HCM. In this study, patients with QTc >480 ms were more obstructive, and there was a weak but significant correlation between QTc and the peak outflow gradient (r^2 =0.05; *P*<0.0001).[14] However, we could not confirm this finding in our HCM population. This discrepancy may be explained by the fact that we excluded HCM patients with a history of surgical myectomy or ablation. We think septal reduction therapy may influence QT interval by subsequent damage to the conduction system and the need for device therapy. Interestingly, Johnson et al[14] reported a higher frequency of myectomy or ablation and cardioverter-defibrillator implantation in patients with a prolonged QTc interval.

There are several case reports that HCM with mid-ventricular obstruction and apical hypertrophy can be associated with ECG changes typical for ST-elevation myocardial infarction (STE and TWI).[15-17] Nevertheless, we found no correlation between these ECG changes with obstructive HCM. Despite the different hemodynamics, the ECG features of OHCM and NOHCM were generally similar. We also conducted a multivariate analysis using a binary logistic analysis for 4 ECG findings with *P* values ≤0.2 and, still, found no independent discriminator. It appears that LVOT obstruction did not produce specific ECG stigmata, and its diagnosis needs echocardiography or pressure measurements during cardiac catheterization.

Conclusion

Despite its unquestionable use in the diagnosis of HCM, no ECG pattern alone or in combination can be used to distinguish between patients with OHCM and NOHCM.

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References

1. Maron BJ. Hypertrophic cardiomyopathy: a systematic review. JAMA 2002;287:1308-1320.

2. Ommen SR, Mital S, Burke MA, Day SM, Deswal A, Elliott P, Evanovich LL, Hung J, Joglar JA, Kantor P, Kimmelstiel C, Kittleson M, Link MS, Maron MS, Martinez MW, Miyake CY, Schaff HV, Semsarian C, Sorajja P. 2020 AHA/ACC Guideline for the Diagnosis and Treatment of Patients With Hypertrophic Cardiomyopathy: A Report of the American College of Cardiology/ American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation 2020;142:e558-e631.
3. Maron MS, Olivotto I, Zenovich AG, Link MS, Pandian NG, Kuvin JT, Nistri S, Cecchi F, Udelson JE, Maron BJ. Hypertrophic cardiomyopathy is predominantly a disease of left ventricular outflow tract obstruction. Circulation 2006;114:2232-2239.
4. Finocchiaro G, Sheikh N, Biagini E, Papadakis M, Maurizi N, Sinagra G, Pelliccia A, Rapezzi C, Sharma S, Olivotto I. The electrocardiogram in the diagnosis and management of patients with hypertrophic cardiomyopathy. Heart Rhythm 2020;17:142-151.
5. Fifer MA, Vlahakes GJ. Management of symptoms in hypertrophic cardiomyopathy. Circulation 2008;117:429-439.
6. Almenar L, Martí S, Roldán I, Miró V, Díez JL, Osa A, Palencia M, Algarra F. Miocardiopatía hipertrófica obstructiva y no obstructiva: diferencias clínicas, electrocardiográficas y ecocardiográficas [Obstructive and non-obstructive hypertrophic cardiomyopathy: clinical, electrocardiographic, and echocardiographic differences]. Rev Esp Cardiol 1996;49:423-431.
7. Corrado D, Pelliccia A, Bjørnstad HH, Vanhees L, Biffi A, Borjesson M, Panhuyzen-Goedkoop N, Deligiannis A, Solberg E, Dugmore D, Mellwig KP, Assanelli D, Delise P, van-Buuren F, Anastasakis A, Heidbuchel H, Hoffmann E, Fagard R, Priori SG, Basso C, Arbustini E, Blomstrom-Lundqvist C, McKenna WJ, Thiene G; Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus Statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. Eur Heart J 2005;26:516-524.
8. McLeod CJ, Ackerman MJ, Nishimura RA, Tajik AJ, Gersh BJ, Ommen SR. Outcome of patients with hypertrophic cardiomyopathy and a normal electrocardiogram. J Am Coll Cardiol 2009;54:229-233.
9. Delcrè SD, Di Donna P, Leuzzi S, Miceli S, Bisi M, Scaglione M, Caponi D, Conte MR, Cecchi F, Olivotto I, Gaita F. Relationship of ECG findings to phenotypic expression in patients with hypertrophic cardiomyopathy: a cardiac magnetic resonance study. Int J Cardiol 2013;167:1038-1045.
10. Rowin EJ, Maron BJ, Appelbaum E, Link MS, Gibson CM, Lesser JR, Haas TS, Udelson JE, Manning WJ, Maron MS. Significance of false negative electrocardiograms in preparticipation screening of athletes for hypertrophic cardiomyopathy. Am J Cardiol 2012;110:1027-1032.
11. Calore C, Melacini P, Pelliccia A, Cianfrocca C, Schiavon M, Di Paolo FM, Bovolato F, Quattrini FM, Basso C, Thiene G, Iliceto S, Corrado D. Prevalence and clinical meaning of isolated increase of QRS voltages in hypertrophic cardiomyopathy versus athlete's heart: relevance to athletic screening. Int J Cardiol 2013;168:4494-4497.
12. Sheikh N, Papadakis M, Panoulas VF, Prakash K, Millar L, Adami P, Zaidi A, Gati S, Wilson M, Carr-White G, Tomé MTE, Behr ER, Sharma S. Comparison of hypertrophic cardiomyopathy in Afro-Caribbean versus white patients in the UK. Heart 2016;102:1797-1804.
13. Panza JA, Maron BJ. Relation of electrocardiographic abnormalities to evolving left ventricular hypertrophy in hypertrophic cardiomyopathy during childhood. Am J Cardiol 1989;63:1258-1265.
14. Johnson JN, Grifoni C, Bos JM, Saber-Ayad M, Ommen SR, Nistri S, Cecchi F, Olivotto I, Ackerman MJ. Prevalence and clinical correlates of QT prolongation in patients with hypertrophic cardiomyopathy. Eur Heart J 2011;32:1114-1120.
15. Cui L, Suo Y, Zhao Y, Li G, Liu T. Mid-Ventricular Obstructive Hypertrophic Cardiomyopathy and Apical Aneurysm Mimicking Acute ST-Elevation Myocardial Infarction. Ann Noninvasive Electrocardiol 2016;21:98-101.
16. Lin CS, Chen CH, Ding PY. Apical hypertrophic cardiomyopathy mimicking acute myocardial infarction. Int J Cardiol 1998;64:305-307.
17. Sawayama T, Nezuo S, Tsuda T, Mizutani K, Harada Y, Samukawa M. A case of hypertrophic obstructive cardiomyopathy simulating myocardial infarction: with special reference to abnormal Q-waves on electrocardiogram (author's transl). Kokyu To Junkan 1981;29:315-321.